



ACC.15

TCT@ACC-12 | innovation in intervention

A887
JACC March 17, 2015
Volume 65, Issue 10S

Heart Failure and Cardiomyopathies

ANDROGEN SUPPRESSION EFFECTS ON HEART FAILURE IN PATIENTS WITH PROSTATE CANCER

Poster Contributions

Poster Hall B1

Saturday, March 14, 2015, 3:45 p.m.-4:30 p.m.

Session Title: Advances in Heart Failure Therapies: From Diuretics to VADs and Transplant

Abstract Category: 14. Heart Failure and Cardiomyopathies: Clinical

Presentation Number: 1146-217

Authors: Zuber Ali, Danielle M. Greer, Robyn Shearer, Andinet Alemu, Arshad Jahangir, Center for Integrative Research on Cardiovascular Aging, Aurora Health Care, Milwaukee, WI, USA

Background: Androgen deprivation therapy (ADT) for prostate cancer (PC) has been associated with adverse cardiovascular outcomes, including heart failure (HF). However, it is unclear whether ADT worsens pre-existing heart conditions or contributes to development of cardiac dysfunction and new-onset HF.

Methods: Patients diagnosed with PC during 2007-13 at a community-based healthcare system were divided into those who underwent ADT with gonadotropin-releasing hormone agonist and 1:1 propensity-matched with patients without ADT. Cox proportional hazards models were developed to examine ADT effects on HF outcomes, including clinical Dx of HF with reduced ejection fraction (HFrEF), HF with preserved EF (HFpEF), low left ventricular EF (<55%), diastolic dysfunction (DD) based on reported grades 1-4 or DD computed from E/A and E/e' echo values. Single- (SM) and multi-variable full (FM) models and stepwise variable selection (SVS) procedures provided estimates of unadjusted and adjusted hazard ratios (HR) and predictors of outcome. Failure rates within 3-yr follow-up were computed using lifetable (actuarial) methods. Men with HF preceding PC diagnosis were excluded.

Results: A total of 1,306 men (653 matched-pairs) were included with mean age of 69.5 yr and a mean follow-up of 3.9 yr. Consistent across SM, FM and SVS, ADT represented a non-significant effect on HFrEF and HFpEF ($P>0.10$) but significantly influenced ventricular function ($P<0.05$). Instantaneous hazard of low LVEF was 3 times greater (SVS adjusted HR: 3.0 [1.2-7.4]; $P=0.01$), but mean number of days to HFrEF was 4,000 d later for ADT (6,900 d) than non-ADT men (2,980 d). Failure rates, respectively, were 0.46 vs. 0.47% at 3 months, 1.9 vs. 1.1% at 1 year, and 6.1 vs. 3.9% at 3 years. Risk of DD was 2 times greater (SVS adjusted HR: 2.0 [1.2-3.3] but mean number of days to HFpEF was 1,500 d later for ADT-treated (6,341 d) than non-treated men (4,800 d). Failure rates, respectively, were 0.6 vs. 0.8% at 3 months, 3.6 vs. 2.2% at 1 year and 14.3 vs. 6.8% at 3 years.

Conclusion: While ADT effects were not evident for HF outcomes based on clinical diagnosis, adverse effects of treatment were detected for systolic and diastolic function based on echo readings.